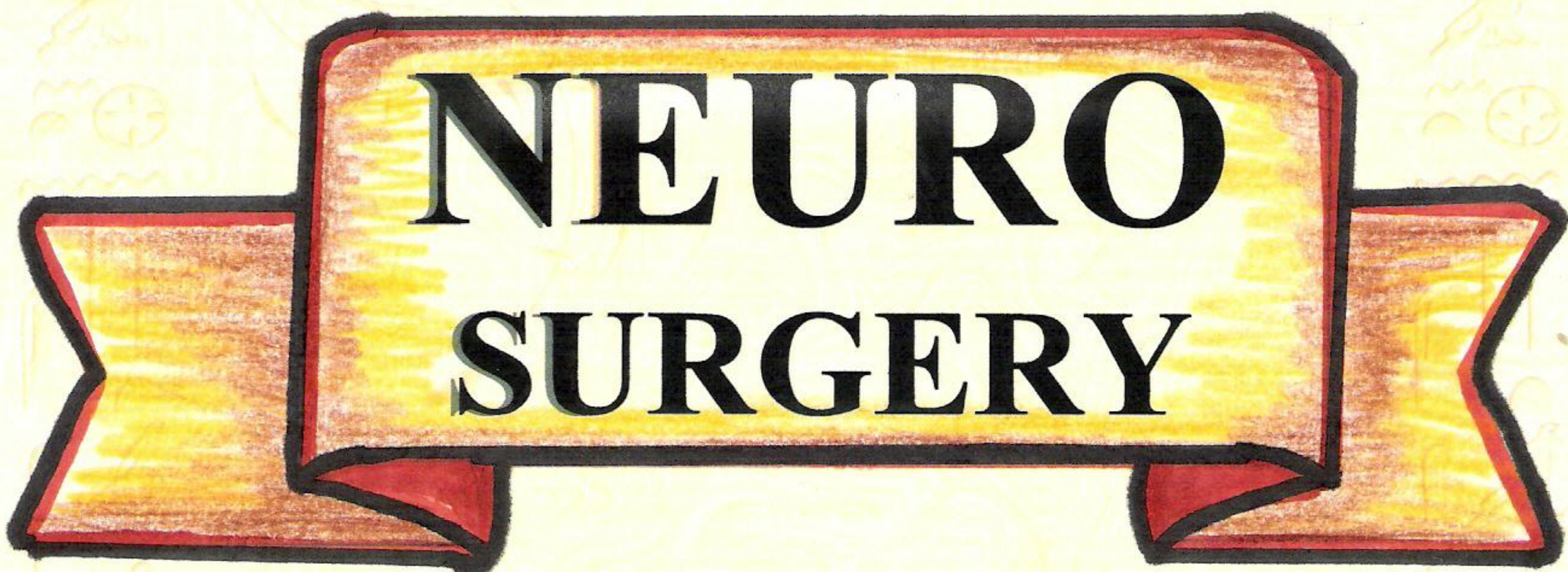




NEURO SURGERY

Dr: K.M.

- 1- Head injury (1)
- 2- Spinal injury (15)
- 3- Peripheral nerve injury (28)

VISIT...

LANZAROTE
Caliente.COM

NeuroSurgery

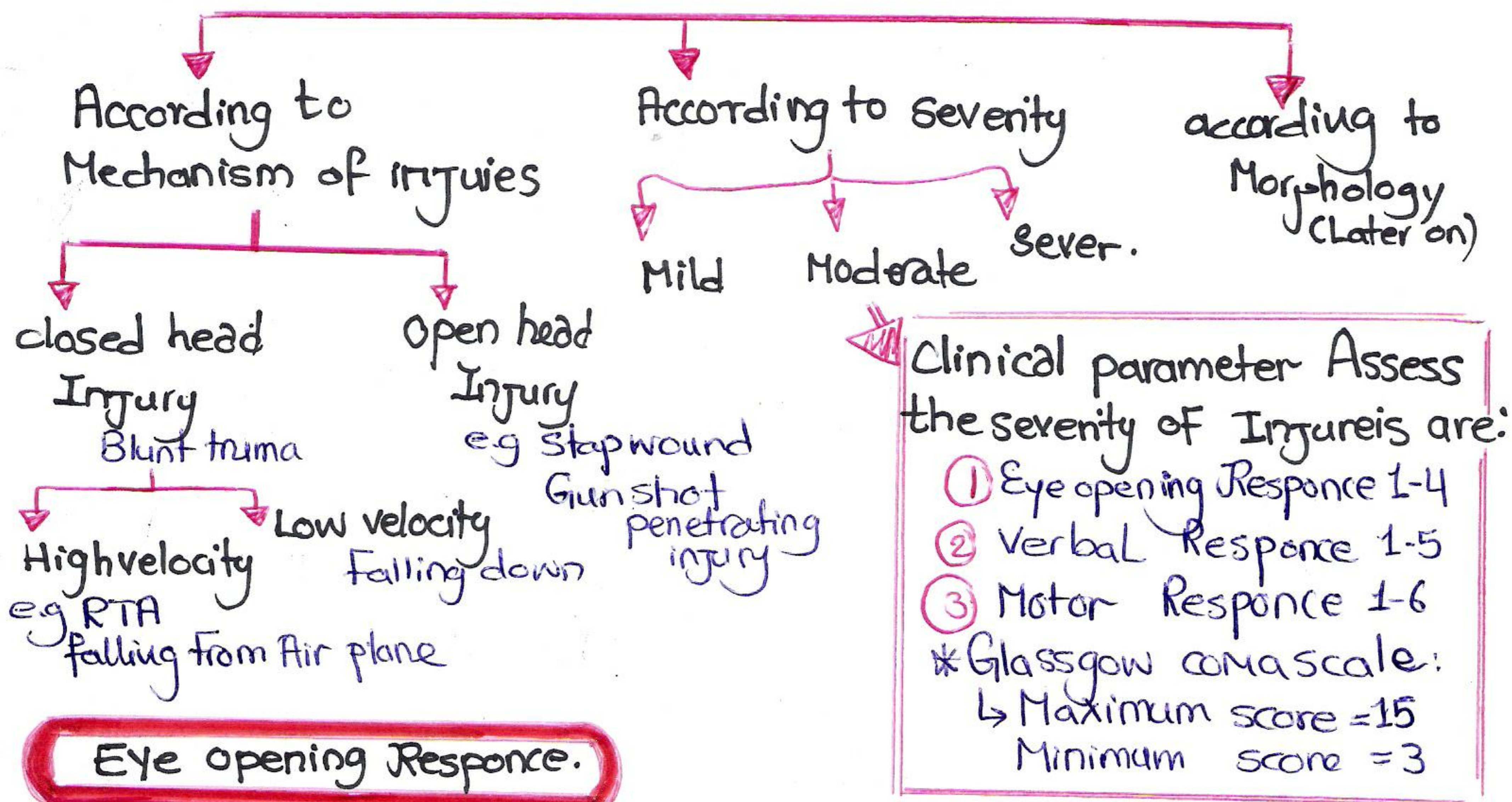
Head injuries

Spinal injuries

peripheral nerve injuries.

Head Injuries

Classification:



Eye opening Response.

- Spontaneous eye opening → 4 score
- opening to call → 3 score
- Eye opening to painful stimuli → 2 score
- No Eye opening (Even with painful stimulus) → 1 score

Verbal Response

- Oriented pt → to time, person, place → 5 score
- disoriented & confused → 4 score
- Inappropriate word → 3 score
- Incomprehensible speech → 2 score
- No Response → 1 score [1]

Motor Response :- the Best

- ① Movement on command → 6
- ② Localized the pain (He try to Remove what's do pain for him) → 5
- ③ Withdrawal Reflex → 4
 - normal Flexion :- (flexion in Hip & Knee, Elbow, Wrist)
- ④ - Abnormal Flexion: occur in pt & decortication damage in cerebral cortex. → 3
- ⑤ Generalized Extension → 2
 - occur in → decerebrated pt → damage of All cerebral Hemisphere.
- ⑥ pt cant move at ~~all~~ whole → 1
 - Flacid paralysis

N.B if the pt move his Rt Limb on command but cant move the Left Limb → His score = 6 on motor Response
 b/c the left limb is Immobile due to Local cause
 e.g: Hemiplegia.

Glasgow Coma Scale :- Higher center function. eg cerebral Hemisphere.
 e.g: pt on punching open his Eye,
 withdraw his limb, & Incomprehensible speech?
 GCS of this pt → $\approx 2 + 4 + 2 = 8$ → Sever Head injury
 ↳ 13-15 → Mild Head injury
 9-12 → Moderate " "
 8 or less → sever " " → Considered as comatose pt

Brain stem Reflex :- Done if GCS indicate sever head injury to assess the prognosis of surviving the pt they include:

- ① size of pupil → Optic Nerve (Afferent) Oculomotor (Efferent)
 (Light Reflex)
- ② corneal Reflex → Trigeminal (Afferent) Facial (Efferent)
- ③ Gag Reflex → Glossopharyngeal (Afferent)
 Vagus (Efferent)

④ Oculovestibular Reflex: Irrigation of ear by:-

NORMAL Response

- Cold water → movement of Eye ball to opposite side associated with Nystagmus.
- Warm water → movement of Eye ball to same side associated with Nystagmus

Cerebral damage
 & Intact Brain stem

→ Tonic Movement of Eye ball (i.e. Nystagmus) to opposite side i.e. cold water & same side i.e. warm water.

Brain stem damage → No Movement of Eye ball

⑤ Oculocephalic Reflex: brisk Movement of Head.

if Eye ball Move to opposite side → Normal brain stem (Normal Response)

if Eye ball Fixed → Move i.e. Head → damage Brain stem...

N.B. Brain death: - Fixed dilated pupil

- Light Reflex → no response
- Corneal Reflex → no response
- Gag Reflex → no response
- GCS = 3
- Apnea test → No Response
- EEG → Flat line

⑥ Apnea Test:-

↑ In concentration of CO_2 (pCO_2) → stimulate Respiratory center → to do it Remove ventilator for 2 min & see Respon.

⑦ EEG if Flat line → Brain death.

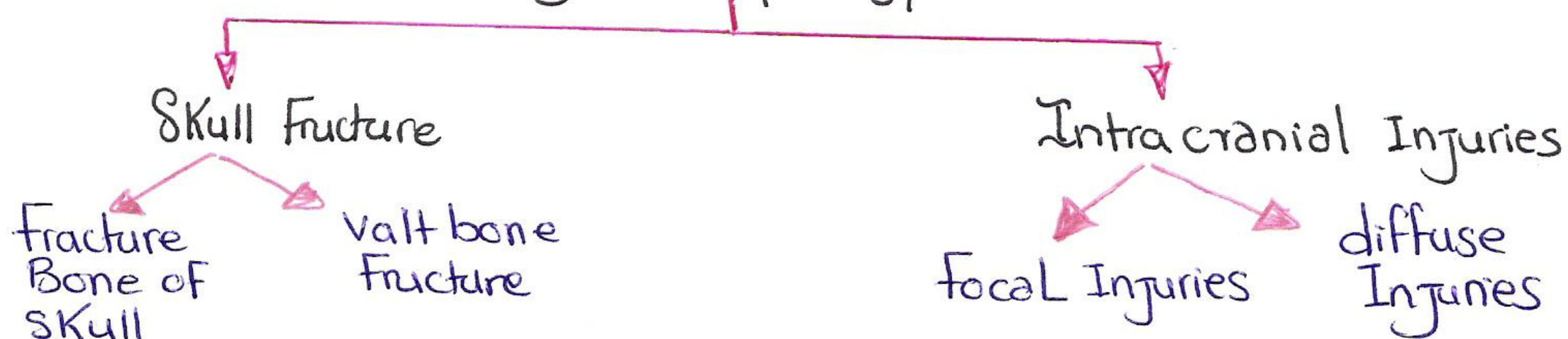
N.B. we can't say Brain death until we do Apnea test & EEG.

- Size of pupil → Isocoria

ANISOCORIA → Emergency b/c Indicate Mass pressure at same side of anisocoria...

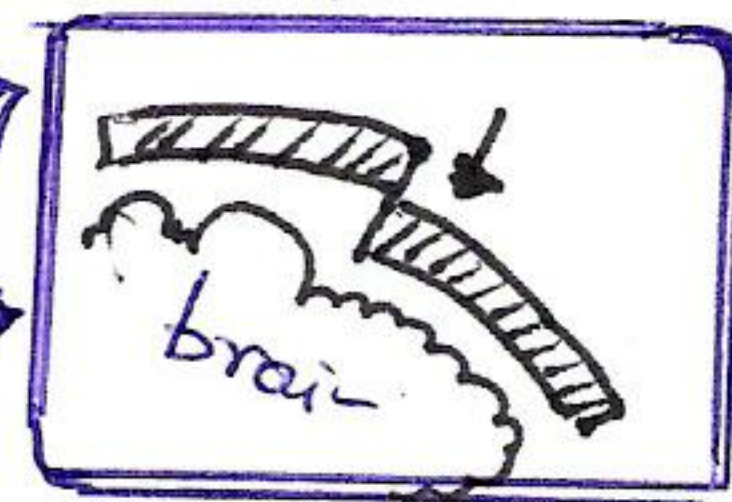
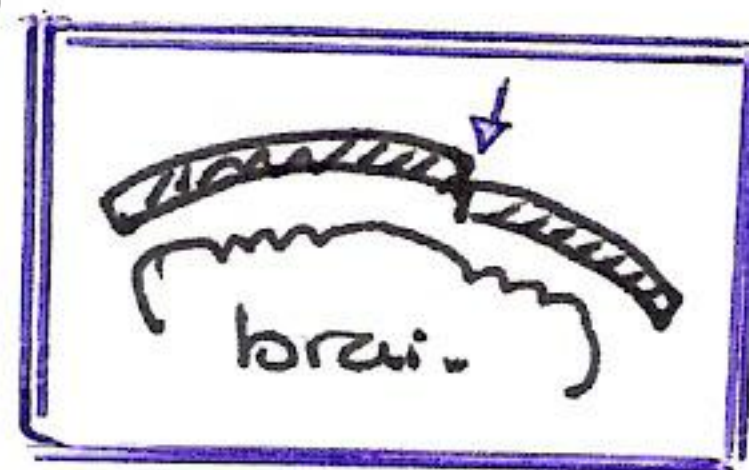
- (4)
- **N.B** Before doing the previous test Brain stem Function
Must we have to Exclude administration of Sedative
• Muscle Relaxants or Hypothermia...

C According to morphology:-



VALT BONE FRACTURE :- May be:

- 1- Linear Fracture:-
- 2- Comminuted Fracture (SATELLITE) :- its Multiple Linear Fracture Radiating from the Impacted area...
- 3- Depressed Fracture :- Dangerous.
 - if the depression is Equal or more than thickness of the skull → surgical intervention. is indicated.
 - if depression Lesser than thickness of skull → NO NEED FOR surgery.



Fracture Base of Skull :- may be:-

- 1- with or without Facial Nerve palsy → ENT surgery
- 2- with or without CSF leak (Lequrrhed)

Clinical Feature of Base of Skull :-

- 1- Nasal discharge → Rhinorrhea → CSF
- 2- Ear discharge → OTorrhea → CSF
- 3- Bilateral periorbital Haematoma → RACCOON EYES.
- 4- Ecchymosis Around Mastoid process → Retromastoid Bruising = Battle's sign
- 5- Bleeding per ear
- 6- facial Nerve palsy.

* Investigation: ① Dipstick strip → For nasal discharge for Glucose (CSF)

② X-ray: to valt fracture (difficult in # Base of Skull)

③ CT: Axial & coronal section → Fracture site of leak

④ Cysternography:- injecting the Dye in CSF and do CTscan → you will see contrast coming out of Site of Leakage.

⑤ MRI: to see if there is leak
*** but NOT to Localize the site of leakage.

Treatment:

① Most cases will Heal spontaneously and leak stop

② prophylaxis Antibiotics.

③ if NOT Resolved For 7 day do Repeated Lumbar puncture
→ ↓ ICP → it is contraindicated in case of High ICP of Any Reson. → Herniation of Brain tissue through Foramen magnum (CONING)

④ Surgical intervention in some case NOT Respond to Repeated L.p.

⑤ if pt come directly e Facial Nerve palsy → Surgery to Repair this palsy → if Develop palsy After 2-3 day → the cause NOT Injury → but developed of oedema. So the pt Treated conservatively...

Intra Cranial Injury

(A) Focal Injury

- Epidural Haematoma (Most common)
- Subdural Haematoma
- Intra Cerebral Haematoma (ICH)

(B) Diffuse Injury

- Mild concussion
- classical concussion
- diffuse Axonal injury

N.B → Type of Intra cranial Haemorrhage :-

- ① Epidural Haemorrhage
 - ② Subdural Haemorrhage
 - ③ Intra Cerebral Haematoma (ICH)
 - ④ Intra ventricular Haemorrhage
 - ⑤ Sub arachnoid Haemorrhage
- } Focal Injury
- } Diffuse injury

* Focal Intracranial Injury :-

- ① Epidural Haematoma: = Extradural Haematoma.
- Def: Collection of Blood Between skull & dura
 - Common site: Temporal & Temporoparietal Region.
 - Bleeding source:
 - 2/3 cases: Middle Meningeal artery & its Branch
 - 1/3 cases: Venous → middle meningeal vein or injury to Venous sinus

* Clinical picture :-

- * primary Loss of consciousness → due to Trauma itself
- then pt Recovery after few Hours → pt develop
- Secondary neurological deterioration → Severe Headache, Vomiting, Blurred vision, Hemiparesis, ↓ level of consciousness, Anisocoria & Even coma. [6]

(7)

N.B - Lucid Interval :- Time between primary loss of consciousness & 2^{ry} Neurological deterioration. or Time Needed by Haematoma to reach Large size to cause Mass Effect,
So it depend ON source of Bleeding → Arterial → cause lucid interval
→ Venous → NO

Venous Bleeding cause small Haematoma → stop bleeding
So In Venous Haematoma → 1^{ry} loss of consciousness Occur
→ 2^{ry} Neurological deterioration NOT occur.

N.B

(A) + Anisocoria → Ipsilateral
+ Hemiparesis → CONTRALateral → Haematoma at site of Anisocoria

(B) + Anisocoria → Ipsilateral → " "
+ Hemiparesis → Ipsilateral

- So the Best clue to determine site of Haematoma is the Anisocoria

NOT Hemiparesis b/c anisocoria Always developed in same side of Haematoma while Hemiparesis may Ipsilateral or contralateral...

N.B - the hemiparesis is CONTRALateral to the damaged area & Ipsilateral to the Haematoma →

False Localizing Sign. → e.g Haematoma on Rt side push Brain stem against Tentorial Edge → damage to cerebral peduncle of left side → Rt side Hemiparesis.

→ Clinical picture in Venous Haematoma

- Haematoma will stop the Bleeding by compressing the vessels.
- Headach without Neurological problem....

- * Diagnosis:
- ① X-ray: fracture of temporal bone
 - ② CT Scan brain + Bone window:- Show
 - Separation of dura from skull (Hyperdense Biconvex Lesion)
 - Midline shift
 - Bone window (CT Bone without brain tissue)
Better to see fracture in skull

N.B → acute Blood in CT → Hyperdense white colour
 subacute Blood in CT → Isodense to Brain tissue
 Chronic Blood in CT → Hypodense (dark in colour)

- Treatment:
- ① if small & stationary → Venous Haematoma → Conservative ttt
 - ② if Large & progressive ↑ in size → Surgery (Craniotomy)

Craniotomy: Evacuation of Haematoma + Stopping Bleeding Source + Return Back the Bone Flap & close the skull

③ if Large & stable → Surgery Craniotomy bc it may cause Epilepsy due to mass pressure effect...

prognosis: if Diagnosed & Treatment Early & properly then Morbidity & Mortality ~~Re~~ Reach Zero...
 if Late diagnosed & Late treated → mortality & morbidity May Reach upto 50%

② Sub-dural Haematoma:
 { Acute
 Subacute
 chronic

- Collection of Blood Between Dura & cerebral tissue...

* Source of Bleeding:- Mainly Venous (Tear in Bridging vein, injury to sinus, Laceration of Brain tissue.)
 * bad prognosis 60-70% Mortality rate...

A Acute subdural Haematoma :

- Clinical picture:
- ① Broad coma or may conscious according to site of Haematoma..
 - ② Brain swelling :- High ↑ Icp due to underlying tissue damage..

Diagnosis :

- ① X-ray: Skull Fracture
- ② CT scan: the haematoma take shape of dura (Sickle) Biconcave Hyperdense lesion with or without mass effect, with or without Midline shift..

Treatment:

- ① Craniotomy and Evacuation of Haematoma
- ② Craniectomy → Remove Bone flap + Evacuation of Haematoma, don't Return Bone Flap again

Advantage of craniectomy

Give More space to Brain for Expansion..
to Stimulate Icp through this Bone window

Bulged Brain → high Icp

Not Bulged brain + Soft Lax brain tissue

prognosis: bad prognosis.

How we can Treat High Icp :

- ① Elevation of Head (45°) → ↑ Venous Return → ↓ Blood amount
- ② Hyperventilation → ↓ pCO₂ to 28 (Normal pCO₂ = 35-45)
→ Cerebral vasospasm → ↓ Blood compartment in brain

③ Drug → (A) Mannitol → it increase osmotic pressure
→ Diuresis → ↓ viscosity of Blood → ↑ cerebral effusion → ↓ production of CSF.

dose.. 1-2g / Kg / day (infusion) it is not give for more than 3 day b/c development of Rebound phenomena.
→ development of edema

* We have Mannitol 5% , 10% , 20% which are are :
ONE we have to use ? - Better 20% b/c of Low Fluid content

(B) Diuretics $\rightarrow$ $\downarrow$ CSF production

(C) Steroid $\rightarrow$ $\downarrow$ cerebral edema.

(d) prophylactic Antiepileptic drugs

(E) sedative & Muscle Relaxant b/c every cough or stress will $\uparrow$ Icp.

(4) Hypothermia: in Sever cases $\rightarrow$ we have $\downarrow$ (Temperature) to 35°C $\rightarrow$ Metabolism in cerebral tissue $\rightarrow$ $\downarrow$ O₂ demand by cerebrum $\rightarrow$ Make it more tolerant to ischemia..

(5) Ventriculostomy $\rightarrow$ (External Ventricular drain) $\rightarrow$ EVD
it is ~~always~~ Not always possible to done
B/c in high Icp $\rightarrow$ Narrow Lateral ventricle
 $\rightarrow$ can't be punctured..

(6) Frontal Lobectomy $\rightarrow$ or Temporal lobectomy (Left side)
these done if there lobe are severely damaged contused or Oedematous it is done unilaterally

- Lumbar puncture

- Indication:

- 1- diagnosis of meningitis , Encephalitis
- 2- diagnosis of subarchnoid Haemorrhage
- 3- Measurement of CSF pressure
- 4- Remove CSF to $\downarrow$ Icp
- 5- Injection of drug & contrast media

Contraindication

- 1- High Icp
- 2- Bleeding Tendency

B- Sub-acute-subdural Haematoma:

acute sub-dural haematoma $\xrightarrow[2-3\text{ wk}]{\text{after}}$ Subacute subdural Haematoma

- Diagnosis :-

ON CT scan: it is isodense to brain tissue so to differentiate it from normal brain tissue, we have to do CT scan $\bar{e}$ CONTRAST $\rightarrow$ show compressed deviated Blood vessels at Haematoma + effacement or displacement of Gyri & sulci

- Treatment: if there is Midline shift $\rightarrow$ (Surgery craniotomy)
if there is No Midline shift $\rightarrow$ conservative ttt.
(midline shift ON CT scan : (mass effect))

Prognosis :- Better than the acute type.

C- Chronic subdural Haematoma.

More common in old age and Alcoholic b/c of:

① Cerebral Atrophy $\rightarrow$ stretching of Bridging vein $\rightarrow$ More liable to bleed even $\bar{e}$ Minor trauma.

② those people more liable to Repeated trauma on head due to More liability to falling down.

Clinical picture :- depend on the site, More common in Cerebral hemisphere.

1- Headach

2- Ataxic Gait (Cerebral Ataxia)

3- Hemiparesis

4- deterioration in level of consciousness

5- Speech may be affected

- Diagnosis: ① CT scan is diagnostic $\rightarrow$ Hypodense lesion dark In colour $\bar{e}$ or $\underline{e}$ Mass effect.

ttt: ① Conservative ttt $\rightarrow$ if NO Mass effect

② if there is Mass effect $\rightarrow$ midline shift.

Surgical Evacuation through simple burrhole (Not craniotomy)
in some cases the chronic Haematoma Septated (capsulated) $\rightarrow$ Need craniotomy to Evacuate.
but Nowaday can evacuate $\rightarrow$ chronic septate subdural hematoma through simple burr hole + Flexible Endoscopy

* prognosis Has Excellent prognosis...

3] Intra cerebral Haematoma.

Rarely caused by Trauma (contusion)
Most common site for contusion → Temporal & frontotemporal, B/c it is Lie directly on skull, but May occur at any place..

Multiple cerebral contusions Hold together with 24 hr → intracerebral Haematoma → called delayed & Traumatic Intracerebral Haematoma.

Diagnosis: -CT Scan:

Show Hyperdense interfere & Hypodense area → Salt & pepper Appearance = not Haemogenous.
Some time hypodense area surround the salt & pepper area (Oedema)

Treatment:

if Haematoma is superficial & cause mass effect
→ do surgery & Evacuate it
if NO mass effect even if superficial → Conservative
if there is NO mass effect + deep Haematoma → Conservative
if there is mass effect &

Diffuse Intracranial Inj

1 Mild Concussion:

pt Has NO History OF Loss OF consciousness
pt Has Neurological deficit (disoriented, confused, hemiparesis)
Recovery completely within few Hour to few day
pt Has NO amnesia

2 classical Concussion:

there is History of loss of consciousness (Not More than 6 hr)
usually 20-30 min $\rightarrow$ if more than 6 hr $\rightarrow$ diffuse AXONAL CONCUSSION

pt has Neurological deficit which Recover within few day to few weeks

there is AMnesia especially ~~from~~ from time of Trauma
but May be Anterograde or postgrade amnesia.

3 Diffuse AXONAL Injury: DAI.

- Loss of consciousness > 6 hr

may be $\rightarrow$ Mild $\rightarrow$ pt Recover from coma within 24 hr

$\rightarrow$ Moderate $\rightarrow$ coma > 24 hr

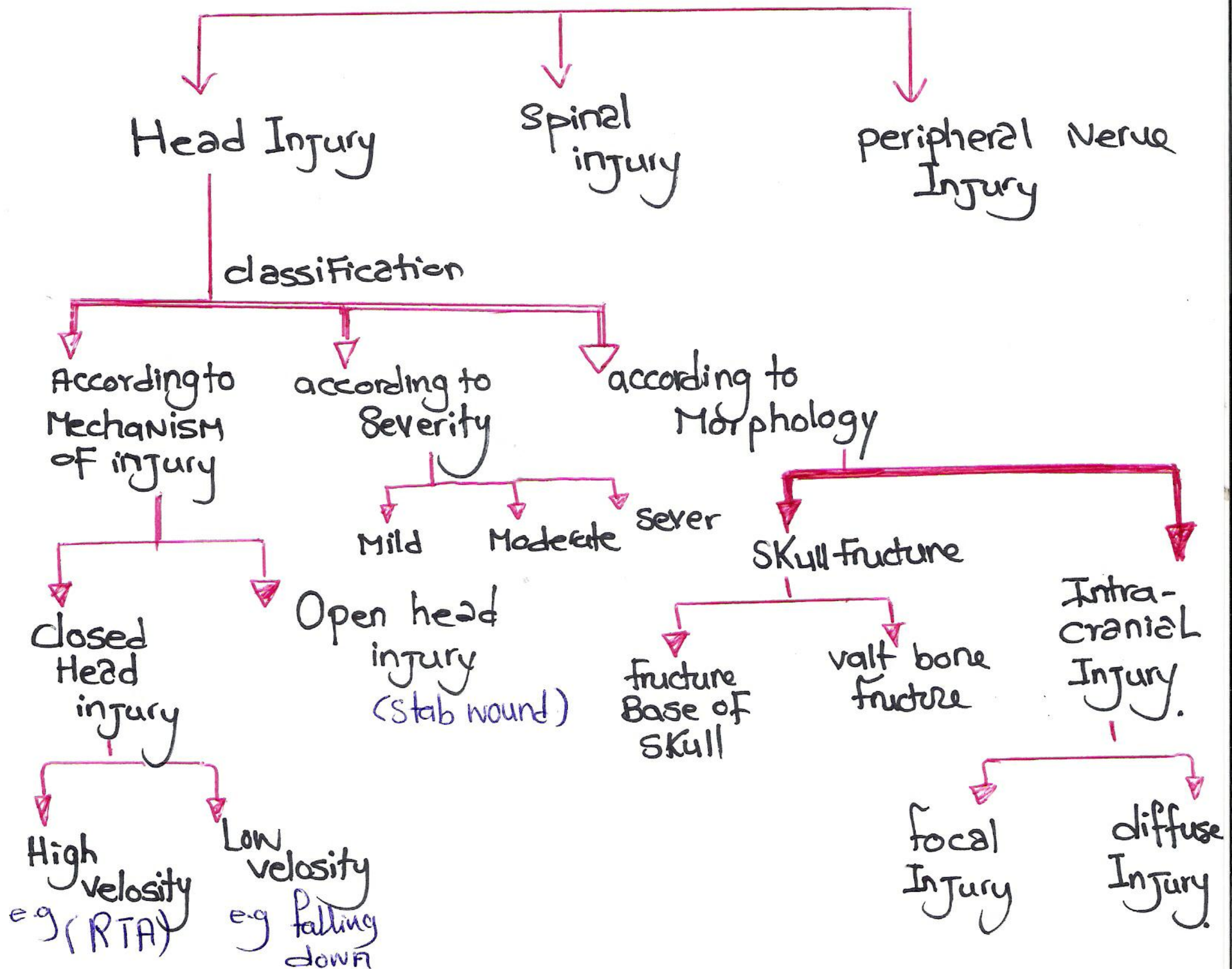
$\rightarrow$ Severe $\rightarrow$ coma > 24 hr + sign of Brain Stem Injury eg Irregular B.p - temperature

... Neurological deficit in mild & Moderate type $\rightarrow$
Will Improved but in Severe it will persist & pt may die

CT scan: usually Normal for DAI (-ve CT scan)

* ttt: conservative management.
Icu Management.

Summary... NeuroSurgery



✶ ✶

Eman G. 2010..

Spinal Injuries

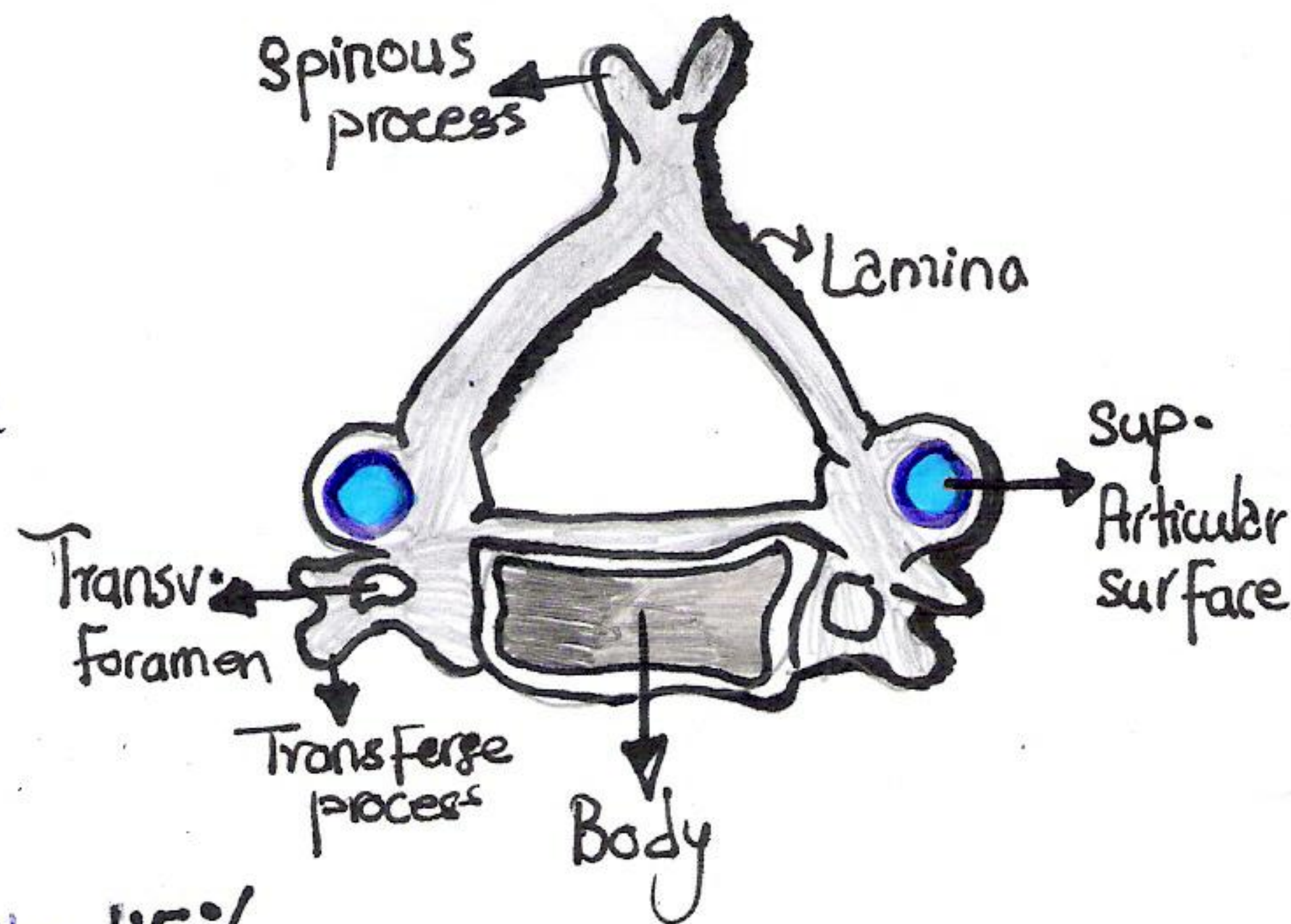
- Site:- → the most part susceptible to trauma is **Cervical** part 60% of Spine Injury..

thoracic spine which is more stable b/c of Ribs & less mobile 8% of spine Injury..

Thoracolumbar Junction 20% of spine Injury (Mobile part)
Lumbar spine 10% of Injury Sacral Region 2% of Injury

** Cervical Spine Inj →

- Incidence
- 60% of spine injury
 - affect 15-30 years/old b/c of car Accident..
 - ♂:♀ 3-4:1



*** Type of Fracture IN General

- * Fracture of vertebral body only 45%
- * Fracture & subluxation 35%
- * ONLY subluxation 8%
- * Traumatic disc prolapse = Fracture or = subluxation & Fracture 10%
- * Spinal cord injury 2%

Type of Cervical spine Fracture

[1] - Atlanto-Occipital dislocation :- Immediate death b/c of Stop Respiration...

[2] - Atlas Fracture (C1 Fracture) :- 3-13% the Most common ONE is comminuted Fracture **Jefferson Fracture**..

Jefferson Fracture Fracture of the ring of C1

usually caused By Axial Loading

the ring can be split into 2, 3 or 4 place.

- Diagnosis :-
- ① Open Mouth view: to Assess displacement:
if the displacement More than 6.9mm
(the Transverse Ligament was Ruptured)
= unstable
 - ② plan X-ray: A-p Film.
 - ③ CT scan.

- ttt :- Halo Jacket for 3 Month → Ensure healing by CT scan.
& flexion Extension X-ray
if Healing fail → pt need posterior occipitocervical
Fusion.

✱ ✱ ✱

③ Axis Fracture (C2 Fracture) :- 18%

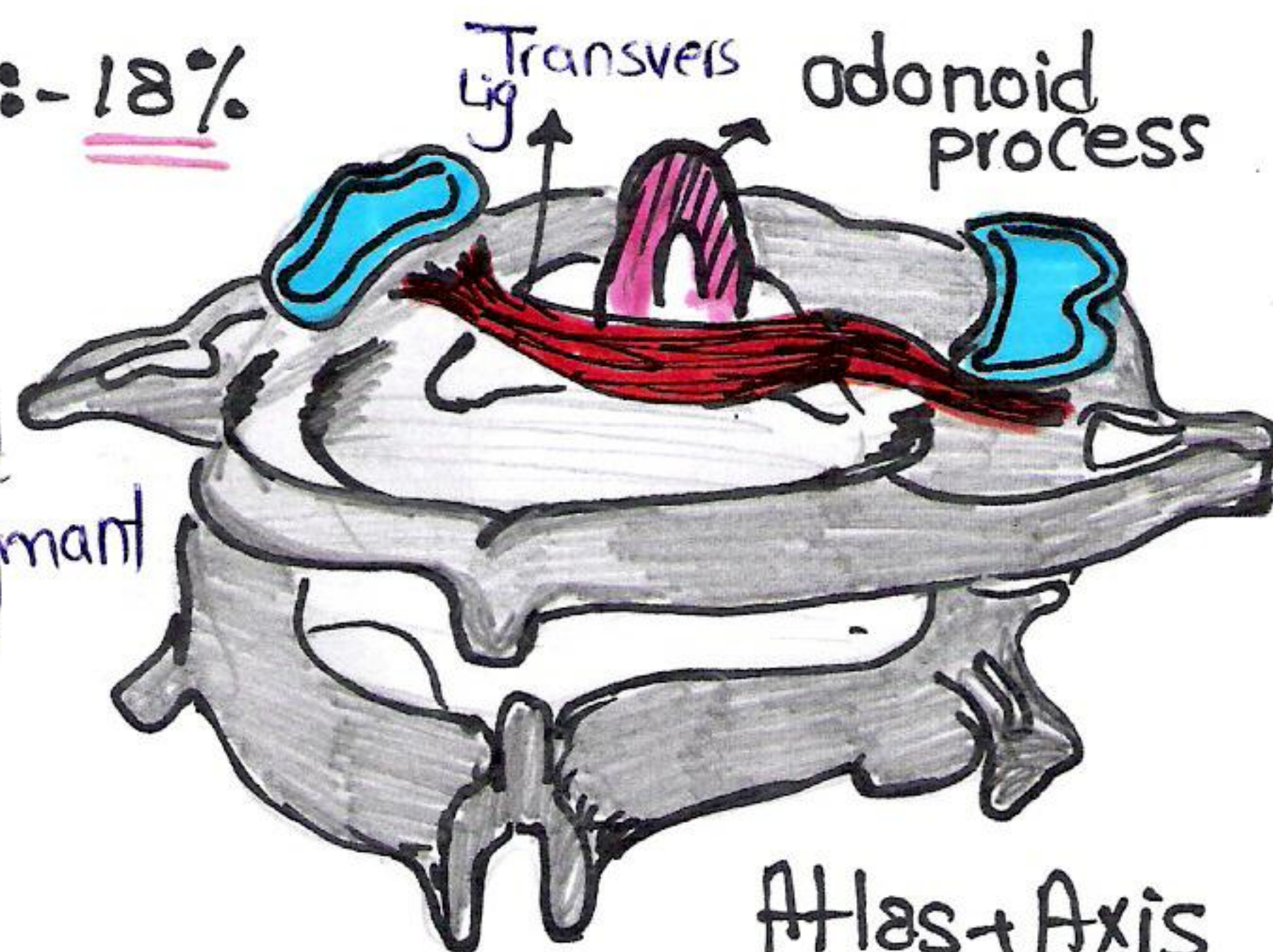
odontoid
process

60%

Hangman's

(pedicle
of C2)

NON
odontoid
NO Hangman's



Atlas + Axis
Articulation.

① Odontoid process # :-

- 60% of Axis Fracture

which is Most commonly Missed Fracture in cervical
Spine & constitute 10% of all cervical injuries...

N.B :- odontoid process :- Lie on Antero superior side
No other vertebra has odontoid process = dens
it is Missing Body of ATLAS B/c it separate from
ATLAS during embryonic development & fuse w
Axis. it project into vertebral column of Atlas.

odontoid - process In child Not fused
6 year & Look's Like Fracture

until

✓ Type of Odontoid Fracture :

① **Type I:** Fracture to Tip of odontoid process
(Rare) usually stable..

can be ttt symptomatically

② **Type II:** Fracture to Base of odontoid process.
70% of displaced fracture → complicated by **NON UNION**

(Undisplaced fracture can be ttt by **Halo Jacket**)
+ **Immobilization** by: Internal fixation (screw) through **ANTERIOR** Approach.

C1-C2 Fusion also used to ttt the condition
Stability should be assessed at End of ttt
with supervised Flexion-Extension Radiography

③ **Type III** :- Fracture to Base of odontoid process
Extending to Body of C2
it heal much Better than type II & Need only
Halo-jacket Immobilization. for 3 MONTH.
Flexion-Extension views should be used for Removing
Jacket to check stability.

② **Hangman's #** .20% of Axis fracture

- Fracture of pedicle of C2
- 5-10% of cervical injuries
- caused by Hyper Extension of spine

→ they are 3 type :-

Type I



Most stable
ttt by Halo-jacket
Immobilization
for 3 Month.

Type II



Most unstable one
ttt by Cervical Fusion
As primary ttt

Type III



In Between

③ **Non odontoid - non Hangman's #** 20% of Axis fractures

ATLANTO-OCIPITAL INSTABILITY

- COMMON IN children.
- Spontaneously due to → Following Trauma or Even due to Infection. in Neck or Oropharynx...

* Clinical picture: usually present as Instability to straighten the Head. which tend to Lock..to one side & slightly up so called: Cock-Robin position.

Diagnosis: ① X-ray
② CTscan E pt looking to Rt & then to left.
« if the position of Axis is Fixed in Relation to the Atlas on two view then → Fixed ATLANTO-AXIAL Rotation can Diagnosed »

* * *

4 C3-C7 Fracture Lower cervical Spine#:

WEDGE #	BURST #	Tear-drop #
MOST COMMON Lower-cervical # caused by: Hyperflexion of Spine ttt: Symptomatic rarely surgical. N.B: stable # & does not cause Neurological damage	caused by Hyperflexion of spine Accompanied by Axial compression. *Fragments of Bone are pushed circumferentially so: spinal cord may be compromised in this type.. of # the # is unstable ttt: IMMOBILIZATION or Surgery (Fusion & Instrumentation)	Fracture + dislocation. When part of injury goes through lower part of vertebra. (& this part may be Ligamentous) Caused by: - (Over Extension. Followed by Over Flexion) # looks Benign in Radiograph but they are unstable..

** FACET Dislocation:

- Caused By Flexion or Flexion with Rotation.
- May Be ONE or Both Facets
- 1 $\frac{2}{3}$ of pt Have Some of Neurological Injury
- 1 $\frac{1}{3}$ of pt Have Complete cord Transection.
- Facet dislocation are unstable. (Need INTERNAL Fixation)

N.B

Anterior disc prolapse Must be Excluded Before reduction.

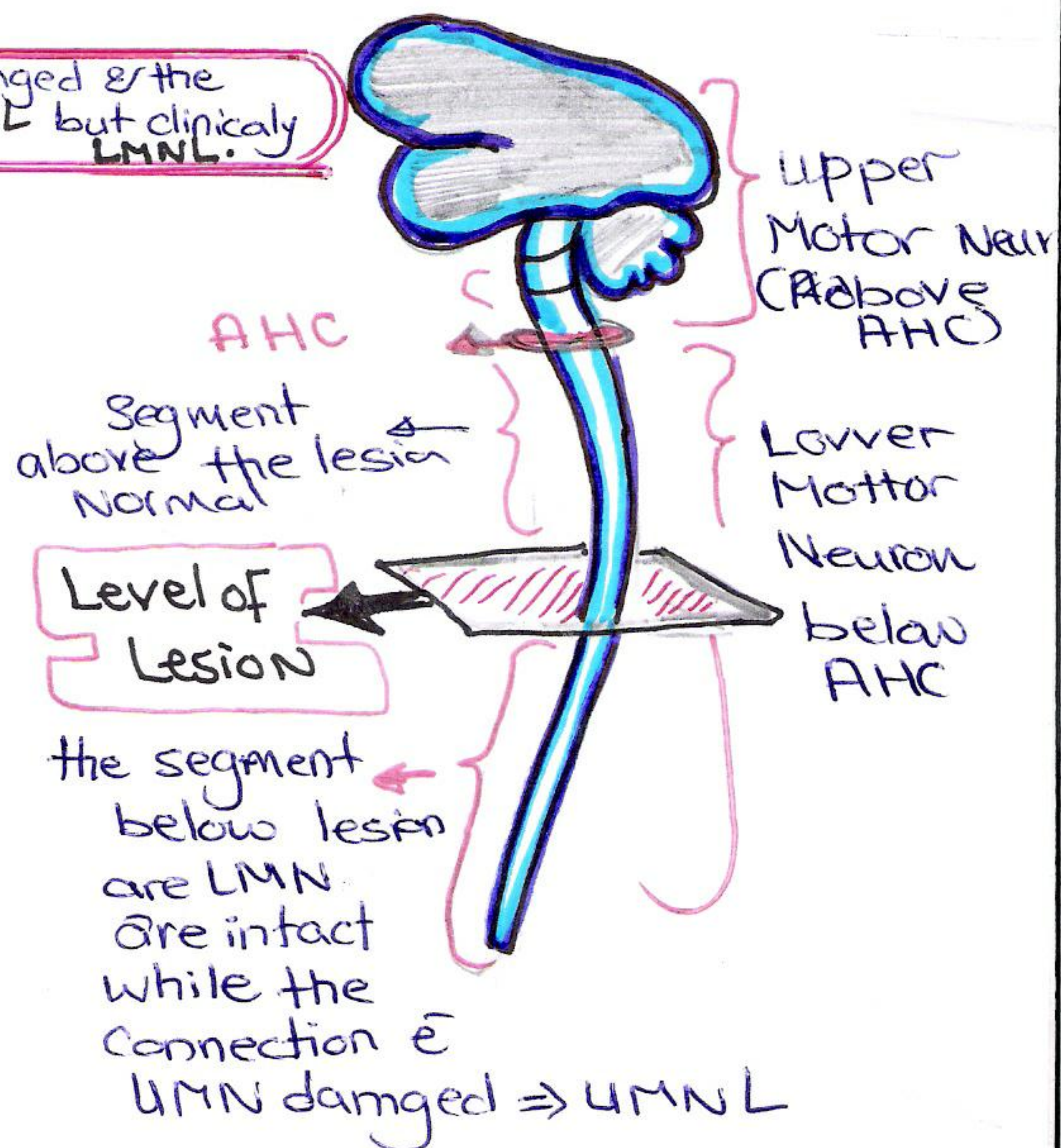
** Clinical picture In cervical spine Injury:-

- depended on site - severity & pre-existing state of pt.
- Complete or Incomplete neurological deficit:
 - UMNL Below the level of injury
 - LMNL At the Level of Injury
 - Normal Neurological function Above Level..

N.B:

at level of the lesion LMNL is damaged & the connection $\in$ UMN $\rightarrow$ LMNL & UMN but clinically LMNL.

	UMNL	LMNL
power	Weakness (pyramidal loss)	Weakness (myotomal loss)
Tone	$\uparrow$ increase	Reduced/Normal
Reflex	prisk	Reduced
wasting	Abscent	present
due to	spinal cord compression	Nerve root compression



N.B:- if complete Neurological deficit $\rightarrow$ $\downarrow$ B.P $\downarrow$ HR, Motor & Sensory & Urinary & Rectal dysfunction.

DIAGNOSIS OF Cerv. Spine Injury

1 HISTORY

- pre-injury Neurological status
- History of the Event
- pre-hospital status & change in neurological status

2 EXAMINATION = Assessment

① Spinal injury (cervical) suspected if :-

there is supra clavicular injury.

head injury / unconsciousness / Facial injury.

High Energy injury → fall from height or high velocity car accident..

Evidence of neurological deficit

Tracheal deviation & Hematoma & swelling in Neck.

Abnormal head position.

pain & Tenderness

② Neurological Assessment:

• power, weakness, Tone

• Reflex change

• Autonomic dysfunction.

③ Assess if the cord Lesion complete or incomplete :-

if the cord Lesion Incomplete the sacral Nerves are those most likely to be spared → **Sacral sparing**

the sacral nerve root are the least affected in

spinal injury probably b/c they are protected to some extent from vascular effects of injury

Sacral sparing can be assessed by :-

① PR Examination to assess rectal sphincter tone.

→ Lax → complete cord injury

→ Tonic → Incomplete

② sacral sensation → Intact → incomplete cord injury

→ Absent → complete " "

incomplete cord injury called =

Brown Sequard Injury

N.B.

IF SPINAL Shock has developed :-

It will not be possible to assess function Below the Injury untill the spinal shock has Resolved at Least Around 24hr, so during this period you cant Assess if the cord lesion is complete or incomplete.

(N.B) • Spinal Shock:

- Occur Immediately After Cord Injury..
- pulse normal + Hypotension
- Flaccid paralysis Including Sphincters.
- Absent Reflexes

Investigation:-

[I] X-ray:

Urgent: → Lateral cervical spine
Chest film - pelvis film
(A-P view)

Non Urgent:

[N.B] Open mouth view is the Best to see → odontoid process & articulation b/w C1-C2.

- Cervical spine
 - A-P view
 - Oblique view
 - Odontoid Tomogram.
 - Open-Mouth view.
- Thoracolumbar view
 - A-P
 - Lateral

[N.B] in Lateral view to cervical spine → 7th cervical spine

Must be in the view (to Make 7th cervical spine vertebra clear in X-ray Either

→ pull Arm of pt toward His thigh to keep shoulder at lower level → Expose 7th cervical vertebra

→ Swimmer view

→ if previous two Technique Not make it clear → do CT scan

- ② CT scan: Best For Bony injury (Fracture), Spinal canal stenosis
↳ Retro-pulsed Fragment...
- ③ three dimensional CT scan: (AXIAL & SAGITAL CT)
Show injury to Bone, Facet, subluxation. More sensitive to bone injury
- ④ MRI: More sensitive to spinal cord & soft tissue injury
e.g. cord compression or contusion.
- ⑤ Myelogram: if there is compression → the contrast will stop & not move..
- ⑥ General Investigation: CBC, ESR - CRP.

MANAGEMENT of cervical spine injury..

- [A] ALWAYS ASSESS THAT THERE IS SPINAL INJURY UNTILL PROVE OTHERWISE..
- [B] Immobilize the spine untill injury has been Excluded to Avoid further injury..
 - Start at site of Trauma - fix head & Neck by attaching.
- [C] if there is associated Life threatening condition e.g. **Shock** you have to deal with it first..
- [D] Fixation & Immobilization :-
 - ① posterior fixation of occiput to upper 3 or 4 cervical vertebra if fracture in ATLAS
 - ② Jefferson Fracture → External fixation.
 - ③ C2
 - Fracture of Odontoid process
 - Type I, III → External fixation
 - Type II difficult & Malunion is common
 - Subluxation:
 - < 5mm → External Fix.
 - > 5mm → open reduction + Internal fixation

- Internal Fixation
 ↗ Anterior Approach by dense screw
 ↘ posterior C1-C2 Fixation.

④ Hangmans fracture: External Fixation (Halo jacket)

⑤ Fracture & subluxation → open reduction + Internal Fixation.
+ Removal of disc (discectomy) and Bone Graft
instead of disc → by time will fuse as one unit.

N.B Disc prolapse should be excluded before reduction is attempted.

⑥ comminuted fracture Bone fragment in spinal cord canal:

Corpectomy: Remove the body of vertebra & put Bone Graft * you have to remove the disc above it & below.
to Allow direct contact Between the Graft & vertebra
Above & below → unit later on.

N.B:

the Most common Body fracture is body of C5
commonest level C5 C6 b/c it is Most Mobile part
in Flexion & Extension..

E Drugs: Steroid: (methyl prednisolone)

Give it directly in Large dose in 1st 8hr (30 mg/Kg)
help recovery ..

N.B

In head injury the steroid used is Dexamethasone
b/c contain few fluid amount while Hydrocortisone
contain Large amount..

prognosis

- • if Complete cord lesion: complete neurological deficit, after 24hr No change to improve him
- • if Incomplete: the earlier management the better prognosis

→ Small number of pt may get progressive deterioration in neurological status In spite of Good Immobilization this occur b/c of persistent compression to cord → interfering w Blood supply to Neurological tissue but may be due to Extension of Haematoma or oedema in area of injury...

N.B:- Trauma & Accident is 1st cause of death before Forties 40y. While After Forties >40y 1st is Heart disease then cancer then stroke then Accident..

N.B Radiological Guideline in spine injury

- A/p diameter of spinal cord
- Countour & alignment of vertebral body
- Bone fragment displacements
- Linear & comminuted fracture
- Soft tissue.

Mechanism of Spine Injury..

- Axial loading → C1 # Jefferson's #
- Hyper Flexion
- Hyper Extension
- Lateral Bending
- Rotation
- Distraction (Hanging)

— ✱ — Eman.G. 2010.
د/ خالد صياد

Thoracic and thoracolumbar Fracture.

(25)

Thoracic fracture T₁-T₉:

- usually NOT common. as thoracic vertebra are supported by Ribs & sternum (rib cage)
- Wedge # Associated @ Osteoporosis are Most common of these & is stable → ttt by pain relief drug + immobilization.
- Other pattern of # in thoracic vertebra, usually associated @ High Energy Multitraumatic injury
- when combined @ sternal #, Aortic rupture, Not uncommon unstable thoracic # are easily Missed on radiograph & can displace.

Thoraco Lumbar fracture T₁₀-L₅

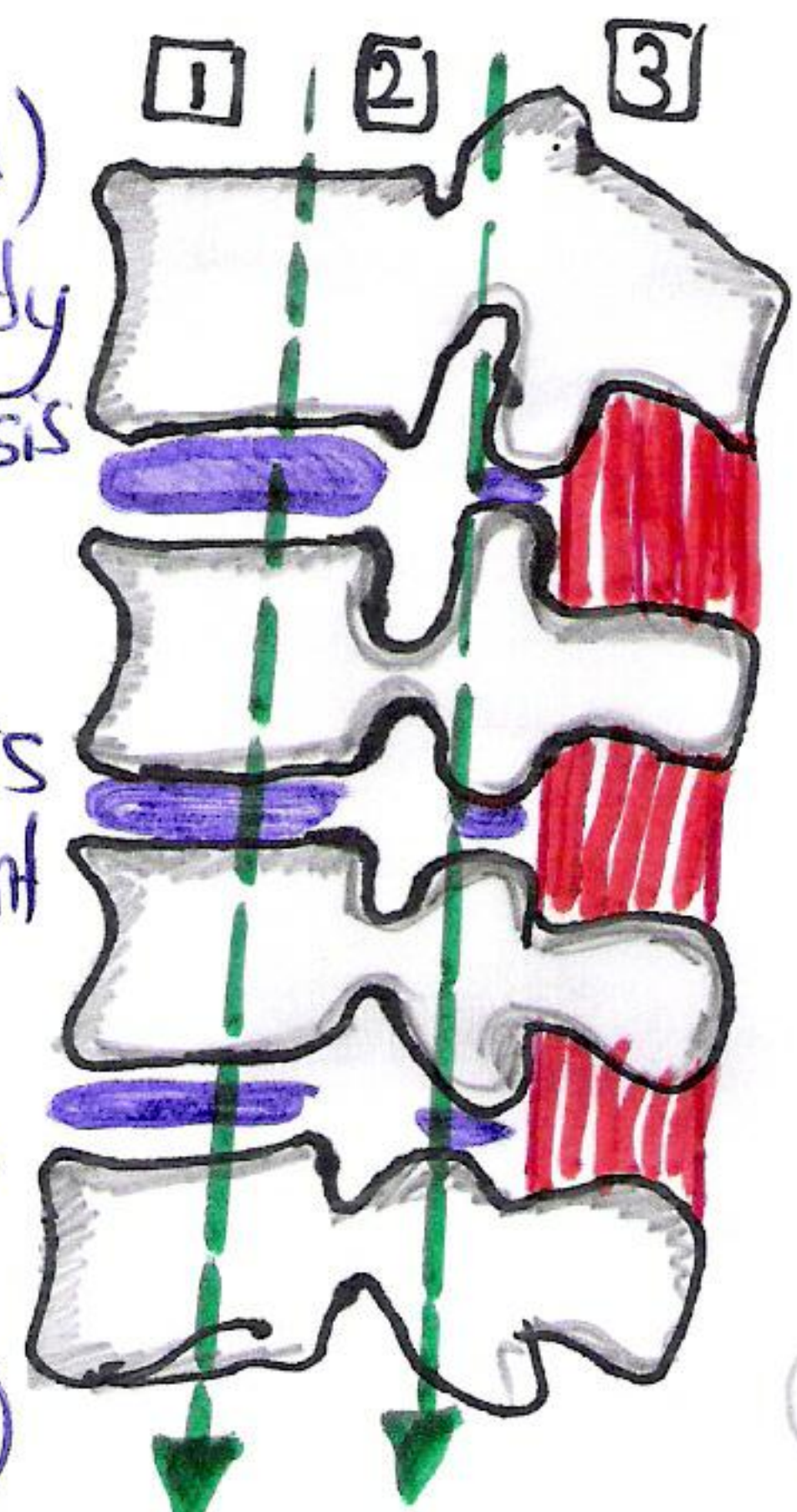
- thoracolumbar # are More common than thoracic # b/c this part of spine not supported by Rib cage
- Most common vertebral # T₁₂ & L₁ b/c these are at junction b/w stiff thoracic spine & Mobile lumbar spine.

*** thoraco-lumbar vertebra are divided into 3 column:

[1] Anterior column: (Ant Longitudinal Liga)
ANT 1/2 of vertebral body
ANT 1/2 of Annulus fibrosis

[2] Middle column: post 1/2 of vertebral bodies
post 1/2 of Annulus fibrosis
post Longitudinal ligament

[3] posterior column: (laminae, pedicle, spinous
ligamentum flavum, facet joint
Inter spinus ligament, Supra spinous ligament)



N.B.:- any Fracture through more than one column is unstable..

Fracture through ONE column is stable fracture & NO need fixation.

Type of Fracture In thoracolum Region.

[1] WEDGE Fracture: (ANT WEDGE Compression)

- **cause:** Flexion Trauma

* Stable wedge* (Most common) Free Neurologically

- **ttt:** ttt Conservative & pain Relieve + Bed Rest
then Mobility & HOLE JACKET.

[2] Burst Fracture:

- **cause:** Vertical trauma. usually it's comminuted *

- **Diagnosis:** X-ray cardinal sign is widening of distance b/w the pedicle.

Fracture in body of vertebra & pedicle → unstable *
if it may be stable.

- **ttt:** in this fracture at least 2 column are affected

ANT + MIDDLE → unstable

Retropulsed Fragment may displaced & compress cord.

*** if pt free Neurologically → 3 things indicated Surgical Intervention:

[1] ANT Height of vertebral body Reduced >50%

[2] Kyphotic Angulation 20-25% (25°)

[3] Spinal canal compromise >50%

(N.B) → 20% spinal canal compromise + Neurological deficit → Surgical intervention..

*** if pt has Neurologically deficit → do surgery

Operation: Transpedicular screw 2 Above & 2 below and do destruction..

if still compression do Corpectomy

3 Splitting Fracture:

(27)

cause: Flexion & Extension (Distraction)

Subtype:- **a** through Bony Elements → • Spinous process
(called chance fracture) • Lamina

b through the ligaments → • Vertebral body
• Supraspinous Ligament
• Interspinous Ligament
• Ligamentum Flavum
• post-longitudinal &
• Intervertebral Ligament

c through bony & ligament

Clinical picture: depend on site of # May be:-

Cord → LMNL

CONUS Medullaris

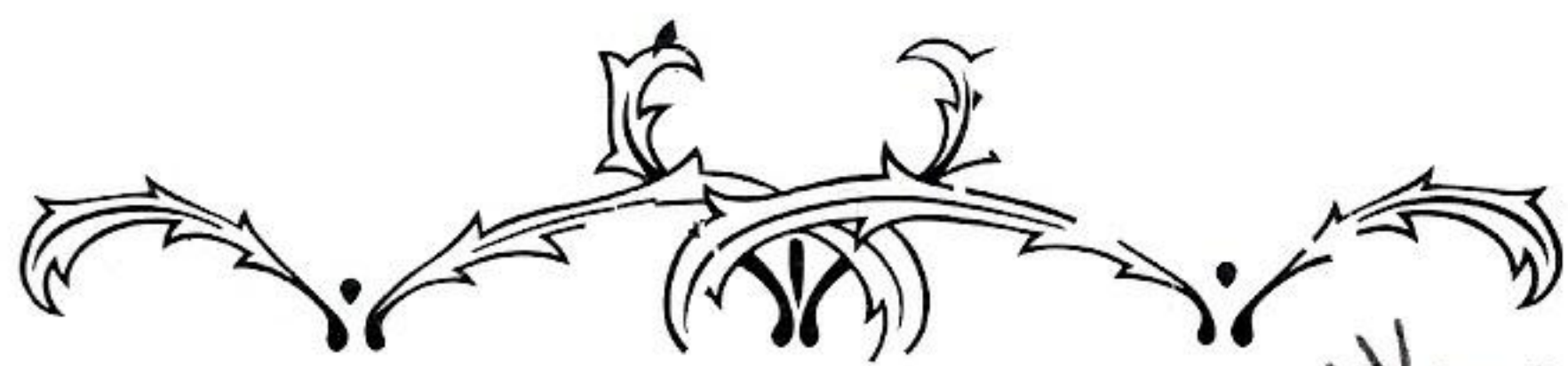
Cauda Equine Roots → more liable to Recovery (LMNL)

* usually Associated w Intra Abdominal Injury

* Splitting # usually unstable b/c it is usually occur through bony & ligament Elements

Diagnosis:- X-ray
- CT scan
- MRI

ttt: posterior fixation by Trans-pedicular screw & decompression.



د/ خالد ميلاد

Eman.G. ☺ / 2010



Peripheral Nerve Injury

* Anatomy:-

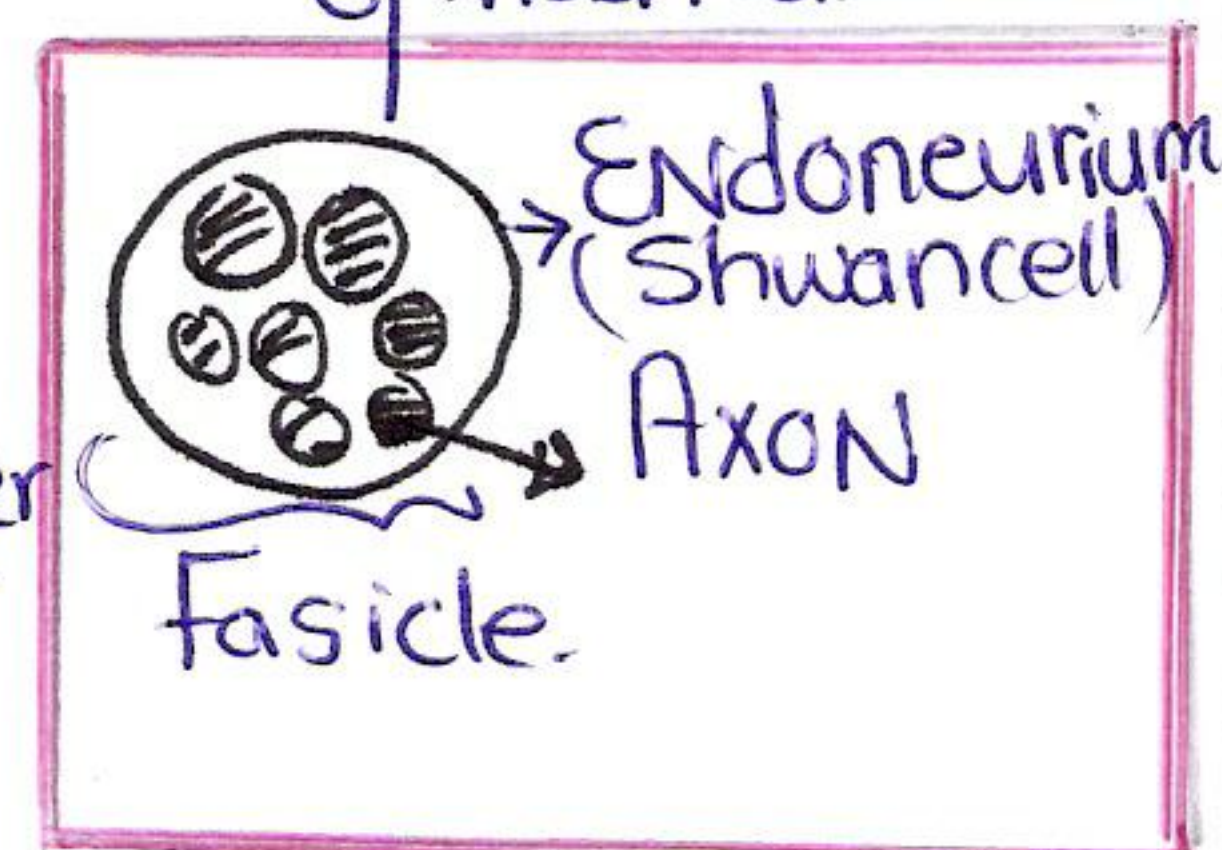
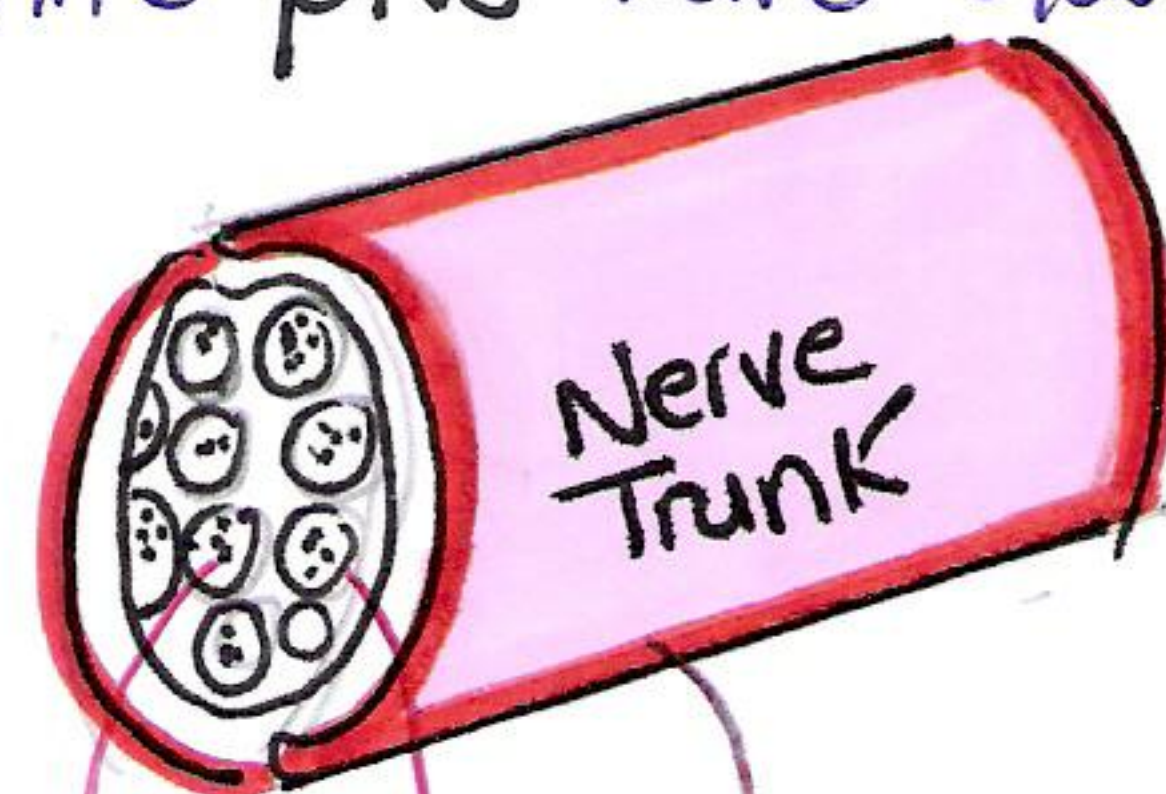
the nervous system divided into Central Nervous system & peripheral Nervous system.

- CNS consist of Brain & spinal cord & 1st two cranial Nerve
- PNS consist of the remaining cranial and spinal cord..
- CNS have ability to regenerate while PNS have Good Ability to Regenerate.



Schwan cell Basement membrane & Endoneural collagen fibers form Endoneural tube.

Large number of ~~fiber~~ Nerve fiber are Gathred in Fascicles surrounded by connective tissue sheet called perineurium, the fascicles are bound together and the whole trunk is surrounded by C.T Layer called Epineurium..



Response of Nerve to Injury

- IF the trauma disrupt the Axon, distal part to injury undergo Wallerian degeneration..

↳ (Lysis of Axoplasm & fragmentation of Myelin sheaths, Leaving an Endoneurial tube containing Schwann cell)

- the Axon In proximal part have the potential to Regenerate Into the Endoneurial tubes of distal segment.

the Regeneration preceeds slowly (1-3 mm/day)

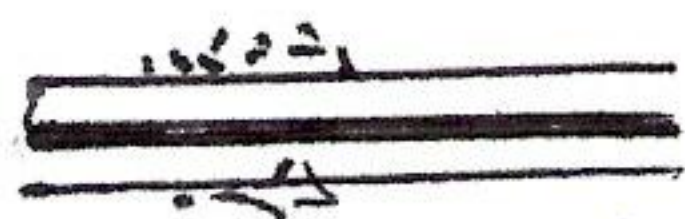
Clinical picture of PNI

- depend on the affected Nerve & severity of injury
- LMNL Flaccid paralysis
- Motor &/or sensory deficit

CLASSIFICATION OF Nerve Injury

SEDDON CLASSIFICATION:

Neuropraxia



- Local block to conduction of Nerve Impulse
 - Axon Intact, continuous
 - No Wallerian degeneration
 - Block is Result from localized demyelination of fibrous in damaged segment of Nerve.
- cause:**
- it is mild injury caused by light pressure (by Tourniquet) or slight stretching of Nerve

ttt:-

No need surgery
complete Recovery
within few day to wk

Axonotmesis

Axon divided



- Anatomical disruption in Axon & there Myeline Sheaths..
- Wallerian degeneration occur in distal part..

cause:

Result from sever blow or stretching injury to Nerve.

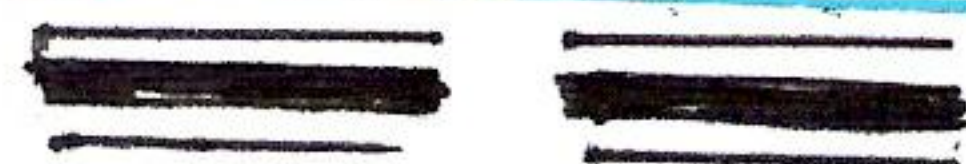
e.g: Radial Nerve palsy associated w/ Humerus (is usually Axonotmesis)

ttt:- Recovery occur by Axon Regeneration (1-3 mm/day) Along the same Endoneurial Tube → Connect to the same Endorgan as before

prognosis: Good
Restoring near Normal Sensory + Motor function.

Neurotmesis

Whole Nerve divided



- Complete cut of Nerve Trunk
- Wallerian degeneration occur in distal part

cause: Result of open Injury as stab wound. Also High Energy traction, Noxious Drug injection & Ischemia can cause it.

ttt: if proper surgical Repair is carried out → Recovery (Axonal Regeneration at rate of (1-3 mm/day))

the quality of Recovery in Nerve perfect. (Rewiring)
this result of failure to correct as Endoneurial tube & other CT have been disrupted Even w/ Best Repair.

Regenerate Nerve fibers connect to muscle or sensory organ w/ did not previously innervate.

[2] SUNDERLANDS CLASSIFICATION

it is classify Nerve injury into 5 degree on the Basis of Increasing Anatomical disruption of Nerve Trunk

Sunderlands classification	AXON	Endoneurial tube	perineurium	Epineurium	Seddon classification
First degree	Intact	Intact	Intact	Intact	Neuropraxia
2nd degree	damaged	Intact	Intact	Intact	Axonotmesis
3rd degree	damaged	damaged	Intact	Intact	Neurotmesis
4th degree	damaged	damaged	damaged	Intact	Neurotmesis
5th degree	damaged	damaged	damaged	damaged	Neurotmesis

this classification used to distinction B/w 3rd, 4th degree inj.
if Fascicles are in continuity (3rd degree) → spontaneous Recovery is possible
but if Fascicles are damaged the spontaneous recovery ---

[1] Grade I (First degree)

- **Cause:** physiological Block at site of Injury
- **C/p:** Light paresis & sensory disturbance
- **Dx:** EMG: Electrophysiological study → **Block At site of Trauma**
- **ttt:** Complete spontaneous Recovery
NO Surgery.

[2] Grade II 2nd degree

- **Cause:-** Axon disruption, Intact Endoneurium
- **C/p:-** Deep sensory & Motor deficit
- **ttt:** Axon Regeneration (1mm/day) the Endoneurial tube act as Guide for Regenerate Axon to the original Target (Excellent Recovery)

3 Grade III (3rd degree)

(31)

cause: Axon disruption, Endoneurial tube disruption

N.B: there is IntraFascicular Bleeding → Fibrosis

→ Interfere ē Regenerating Axon to proceed →

No Excellent Recovery

(Recovery depend on Extend of Fibrosis)

if Fibrosis Mild to Moderate:-

Regeneration 60-80%

- Recovery of Function B/c there is No Guide for Regenerating Axon.

if Sever Fibrosis:-

interfere ē

Regeneration ..

Need Surgical Intervention.

4 Grade IV (4th degree)

Cause: Axon, Endoneurium/peri erium are disrupted.

No spontaneous Recovery → Need Surgry.

5 Grad V 5th degree

Cause: Complete cut of Nerve trunk.

No chance of Recovery, Need Surgry.

	Neuropraxia	AXONotemesis	Neurotmesis
① Motor Loss	Complete loss	complete loss	complete loss
② Sensory loss	partial sparing	complete loss	complete loss
③ Autonomic function..	Spared	Lost	Lost
④ Nerve conduction distal to injury..	present	Abscent	Abscent
⑤ Recovery	Rapid & complete 3mm/day	Good 1mm/day	Always Imperfect (1mm/day)

DIAGNOSIS OF peripheral Nerve Injury

32

- ① history of Trauma
- ② clinical Examination:-
 - ⓐ Motor Function → Weakness / Power
 - ⓑ Sensory Function → Two point discrimination (very sensitive)
 - ⓒ Autonomic Function → Loss of sweating
- ③ X-ray:- for Exclude Fracture of Bone Related to Nerve Injury.
- ④ MRI: if there is Evulsion in Nerve root → as by pseudomeningocele (cyst)
- ⑤ Electrophysiological studies = Neurophysiological study.
it is Main Diagnostic Test
we Must wait 2-3 wks After Nerve Injury
before these Test can be performed b/c within 2-3 wk of damaged Nerve has Normal Electrophys-Study as demyelination = Wallerian degeneration
Need 1 wk - 10 day to occur..

→ Type of Electrophysiological Study → EMG / SSEP

① EMG: Electromyography: muscle action potential are Recorded in Response to voluntary Activity

② SSEP: (Somatosensory Evoked potential) :-

Nerve conduction Study Involve Recording of Sensory or Motor Nerve Action potential & calculating velocity for Given Anatomical segments → slowly conduction (Abnormalities)



CAUSE (MECHANISM) OF PNI

- ① Laceration injury (Stab wound)
- ② Contusion injury (blunt trauma)
- ③ Compression injury → Carpal Tunnel syndrome.
Disc prolapse.
Compartment syndrome.
Tight plaster.
- ④ Stretch & Evulsion injury → Motorcycle Accident
Erb's palsy.
- ⑤ Extrem Temperature Either Extrem cold, Extreme heat
Burn.
- ⑥ Injection injury → Toxin, chemical → Neuritis.
- ⑦ Electricity.

TREATMENT OF PNI. peripheral Nerve Injury

Blunt Injury

do EMG after 2-3wk of Injury → don't do immediately after trauma but wait 2-3wk b/c before it Normal EMG
Even if there is complete Nerve cut as demyelination Wallenian degeneration (occur within 1-10 day)
demyelinated Nerve → Abnormal EMG
NON demyelinated Nerve → Normal EMG

Laceration Injury.

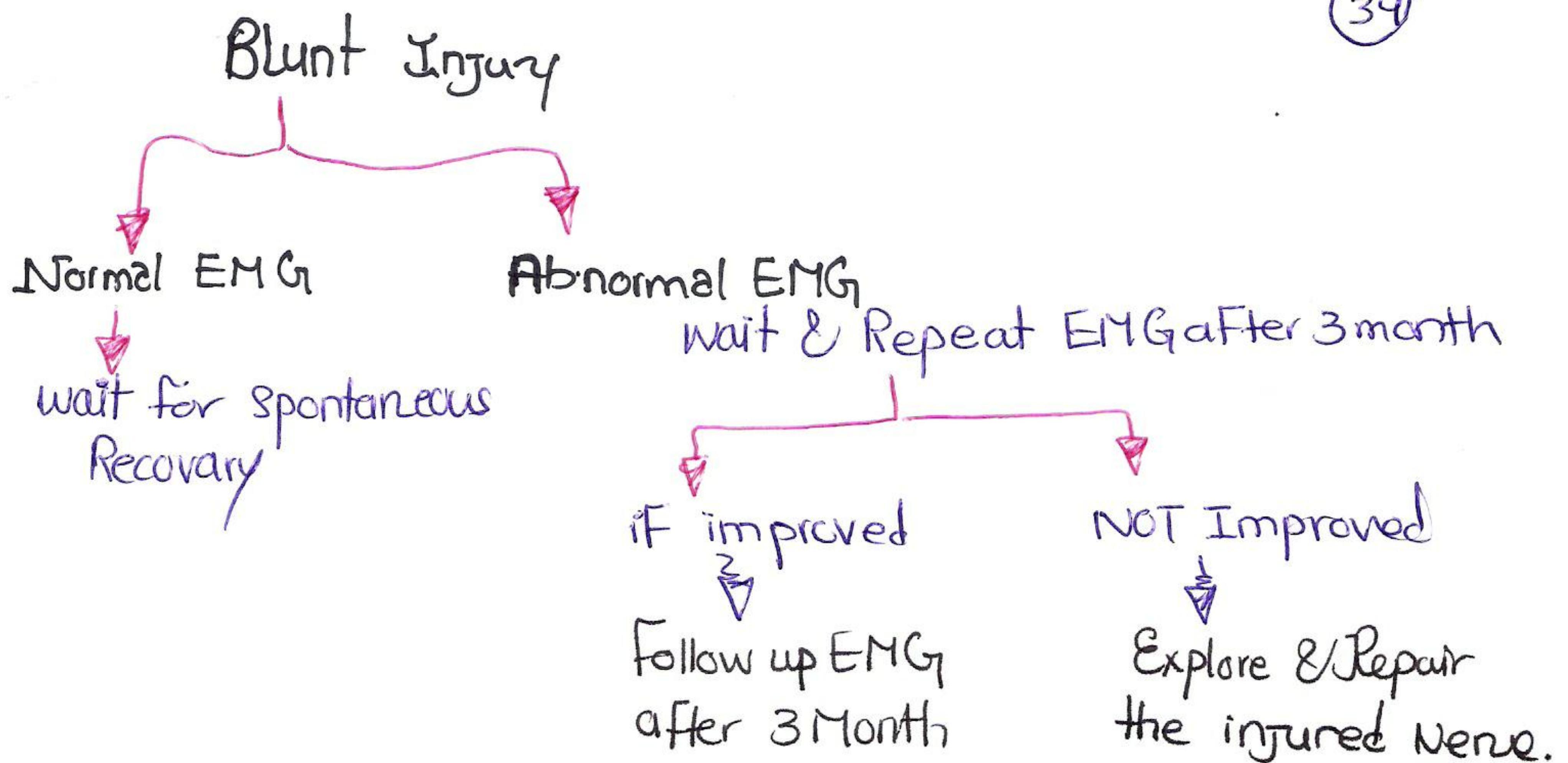
No Contusion

primary Repair

Contusion occur

Secondary Repair.

wait 10 day - 2wk until odema subside then Remove Contused part of Nerve.



Prognosis ..

* Factor Affecting Recovery :-

- ① Age : Children recovery better than Adult
- ② Severity: Clean cut do best → High energy Injury e.g (Gunshot)
(damage of Nerve & has worse prognosis)
- ③ Level: Distal Lesion do Better than proximal
b/c the distal Lesion Need less time to Recover the Length of part distal to Lesion (1-3 mm/day)
- ④ Better In pt without premedical disease (DM, Hypertension) & in NON SMOKER better than SMOKER.
- ⑤ Type: Motor Nerve to Large Muscle do Best
- ⑥ Other injuries: Soft tissue damage, vascular injury & fracture make the prognosis bad.
- ⑦ delay: the Earlier repair the Better prognosis.

N.B: why prognosis In distal Lesion Better than proximal?

- ① - proximal need more time to Recover.
- ② - proximal Lesion the Motor & sensory fibers are present in same trunks while In distal the sensory & Motor are separate. → sensory & Motor pathway confused with Each other...

- Follow up:-

- ① by EMG → More sensitive.
- ② Clinically → Observe proximal Muscle to injury. (Examine it is functioning) & calculate time needed to become functioning (Reinnervated) ($1-3 \text{ mm/day}$) → if time needed correspond to the distance B/w the Muscle & site of Injury → Good prognosis..

Clinically Examine Reinnervated Muscle by Tapping on the muscle

- if there is Reinnervation there is fasciculation of Muscle
- if No Reinnervation → No fasciculation.



Eman.G. :)
2020

CARPAL Tunnel Syndrome

** Compression Injury

* Incidences: More common in ♀ > ♂ (7:3) b/c the Carpal tunnel in women is narrower than man

* predisposing Factor:

- ① Obesity
- ② thyroid disease
- ③ Malunion of fracture in this area
- ④ Contraceptive pills
- ⑤ Acromegaly
- ⑥ RA Arthritis
- ⑦ wrist trauma
- ⑧ Exostosis
- ⑨ Dialysis & chronic Renal Failure

* Boundaries of CARPAL Tunnel:

ANTERIOR → Flexor Retinaculum

POSTERIOR → carpal Bone

* CONTENT :- Median Nerve, Flexor Tendon of Finger.

* Clinical picture:

- ① parasthesia (lateral 3 1/2 Finger)
- ② pain (Night > daytime)
- ③ weakness & Atrophy of Thenar Muscle.
- ④ falling object: weakness of Hand Grip!

burning sensation pins & Needle Removed by Shaking Hand

ASSESSMENT:

- ① Tapping → parasthesia + pain
- ② phalen test → flexion of wrist 60/second +ve parasthesia

② Diagnosis

- ① EMG
- ② SSEP
- ③ CT Scan

** Treatment:

① Mild

- * Antiinflammatory drugs
- * support the arm
- physiotherapy

② Moderate to severe

- * Surgical decompression of Median nerve. Either by :-
 - ① classical opening Local anaesthesia
 - ② Endoscopic surgery
- Better B/c there is NO Fibrosis → NO Recurrence.
- * Insizing the Flexor Retinaculum.



بالشوق...
Eman.G. 😊 / 2010.